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UPPER GASTROINTESTINAL BLEEDING — STATE OF THE ART

Abstract: Upper gastrointestinal (GI) bleeding is a condition requiring immediate medical intervention, with high associated mortality exceeding 10%. The most common cause of upper GI bleeding is peptic ulcer disease, which largely corresponds to the intake of NSAIDs and *Helicobacter pylori* infection. Endoscopy is the essential tool for the diagnosis and treatment of active upper GI hemorrhage. Endoscopic therapy together with proton pump inhibitors and eradication of *Helicobacter pylori* significantly reduces rebleeding rates, mortality and number of emergency surgical interventions.
This paper presents contemporary data on the diagnosis and treatment of upper gastrointestinal bleeding.

Key words: endoscopy, upper GI bleeding.

INTRODUCTION

Obtaining data on the incidence of upper GI bleeding in Poland and the number of hospitalizations for this reason is complex. Categorization of diseases according to ICD-10 catalog extracts some diseases that cause GI bleeding; however system of reimbursement of medical expenses based on Homogeneous Groups of Patients (HGP) often leads to a distortion of these data. According to the data obtained this way there were 31,927 patients in 2009 and 27,280 patients in 2013 hospitalized due to GI bleeding and categorized into HGP F62 (Large and therapeutic endoscopic procedures in GI bleeding), F63 (Mid and diagnostic endoscopic procedures in GI bleeding) and F66 (GI bleeding — conservative treatment) [1].

The summary of these data suggest that the incidence of GI bleeding in Poland and other countries decreases, affecting 83/100000 inhabitants in 2009 and only approximately 72/100,000 inhabitants in 2013. No change in ratios within morbidity in women (40%) and men (60%) has been observed. In the US, upper GI bleedings account for 400,000 hospitalizations per year with the total hospital costs and work absence reaching nearly 2 trillion dollars [2]. The incidence of hospitalization for GI bleeding is steadily decreasing [2–5]. The etiology of bleeding ulcer is associated with increasing use of non-steroidal anti-inflammatory drugs

(NSAIDs) and *Helicobacter pylori* infection [1, 2]. Men are twice as likely to have GI bleeding, and its incidence increases with the age of patients. Recurrence of bleeding occurs in about 15% of hospitalized patients and is associated with more than 10% mortality [1, 2, 6].

ETIOLOGY

Upper GI bleeding, which source is located from the esophagus to the ligament of Treitz is caused in 50–70% of cases by gastric and duodenal ulcer [1–3, 5, 6]. Other cases are usually related to hemorrhagic gastritis and duodenitis (10–20%), esophageal varices (3–10%), Mallory-Weiss tear (3–7%), malignancy (approx. 2%) and vascular malformations (2–10%). In a few percent of cases one fails to recognize the source of bleeding [1, 7] (Table 1).

Table 1

Common causes of upper gastrointestinal hemorrhage.

Etiology	Incidence (%)	Mortality (%)
Peptic ulcer	20–60	4–6
Erosions	7–30	0–1
Varices	4–20	30–50
Esophagitis	3–15	0–1
Mallory-Weiss	4–15	0–2
Vascular malformations	2–5	
Malignancies	1–5	20–40
Others	<2	2–8
Not known	<3	

Duodenal peptic ulcers occur more frequently (approximately 55%), but are less likely the cause of hospitalization (38%). According to American sources, mortality caused by bleeding from duodenal and gastric ulcers is 3.7% and 2.1%, respectively. The reason for this is the proximity of the posterior wall of the duodenum with large vessels and hence, more dynamic bleeding [1, 8].

Peptic ulcer is a distinct breach in the mucosal lining of the GI tract which penetrates through the muscularis mucosae and lamina propria (Fig. 1, Fig. 2). This mucosal defect is accompanied by inflammatory infiltration with deeper tissue necrosis. Local progressive necrosis may involve arterial and venous walls, causing interruption of their continuity with subsequent bleeding. Peptic ulcer may bleed from the surface or edges of the ulcer bed, but also from a vessel within its niche. A meta-analysis of 16 studies involving more than 1600 patients

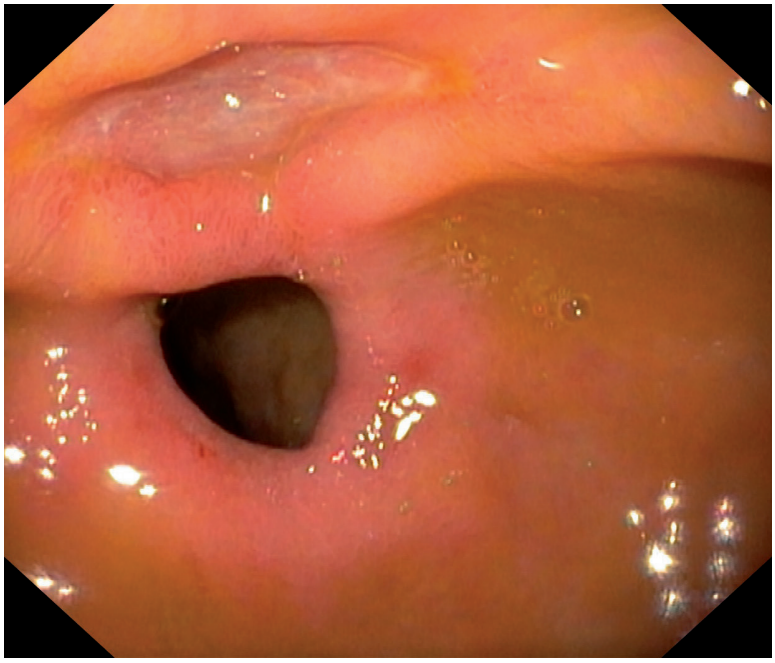


Fig. 1. Gastric ulcer.

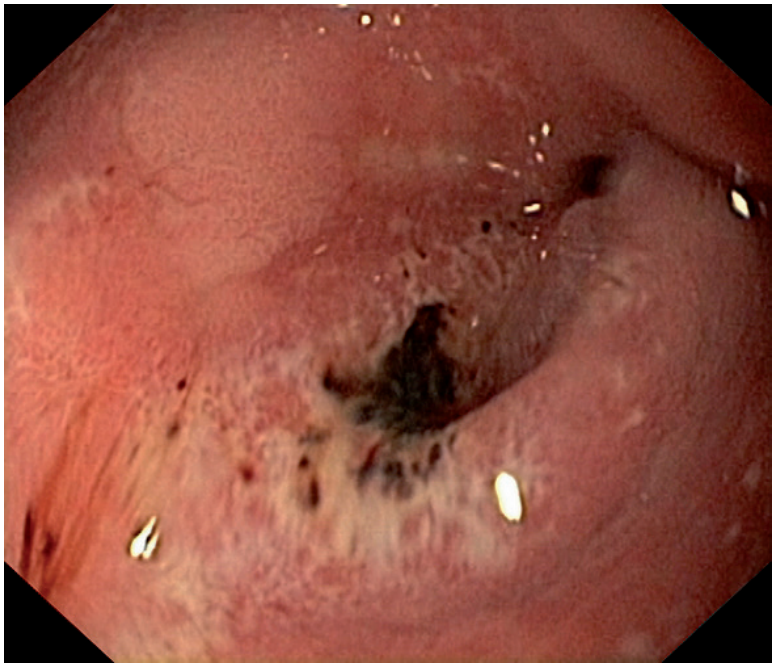


Fig. 2. Duodenal ulcer covered with a clot.

taking NSAIDs showed that the additional *Helicobacter pylori* infection increases the risk of GI bleeding (OR = 6.1) [9]. The bacteria adhering to the mucosa make it more vulnerable to the action of enzymes and toxins, which results in increased levels of gastrin and acid concentration in the stomach.

Erosions are shallow epithelial defects and a symptom of acute mucosal inflammation. The inflammatory process can appear as acute hemorrhagic gastritis or duodenitis (Fig. 3). It reveals the presence of numerous reddish spots that are bleeding or covered with small clots. The factors that cause this inflammation may be chemicals (including drugs and alcohol), bacteria, toxins, stress, and certain neurological diseases [2]. The extent of pathologic changes depends on the type of inducing agent, its amount and concentration and duration of action.

Esophageal varices are running superficially in the mucosa and submucosa dilated veins.

These venous dilations occur due to a medical condition located outside the gastrointestinal tract and are usually discussed as a separate disease entity [2, 10]. Esophageal varices are a major complication of portal hypertension which is most frequently the result of liver cirrhosis. Blood flow through the hepatic portal system is then redirected from the liver into areas with lower venous pressures. This means that collateral circulation develops in the lower esophagus and stomach draining blood to azygos vein. The small blood vessels in these areas become distended, becoming more thin-walled, and appear as varicosities. A sudden rise in venous pressure combined with a weakening of the walls to stretching, evokes variceal rupture and hemorrhage. On endoscopy varices appear as swollen veins with one or more prominent cherry-red spots from which blood flows in a continuous stream. Varices collapse after bleeding and hence it is hard to accurately locate the site of variceal rupture.

The longitudinal rupture of the mucosal lining in the lower part of the esophagus at the junction with gastric cardia is referred to as Mallory-Weiss tear (Fig. 4). Damage to the wall may reach the submucosa, muscularis propria, and in exceptional cases involve its full thickness leading to mediastinitis. Bleeding occurs most frequently at the end of vomiting, and its intensity depends on the size of the disrupted vessel, and the amount and depth of the mucosal breaks [3, 5, 11]. A clot located in the area of gastric cardia with a tiny blood leakage underneath is the most common endoscopic image of such a tear.

Esophagitis is evoked by gastroesophageal reflux disease, infection, drugs, radiation and burns [2] (Fig. 5). It frequently involves the lower portion of the esophagus, and usually does not require endoscopic intervention.

Neoplastic infiltration is the most common cause of slowly progressive anemization related to minor surface damage of the infiltrate with macroscopically inconspicuous bleeding (Fig. 6). The intensity of bleeding from neoplastic infiltrate depends on its extent, diameter and number of damaged vessels. Endoscopic

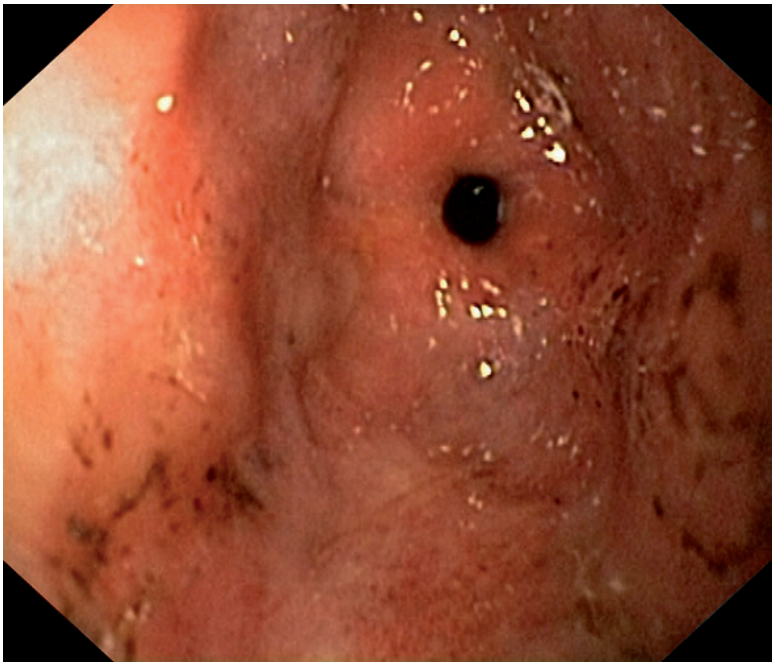


Fig. 3. Hemorrhagic gastric erosions.

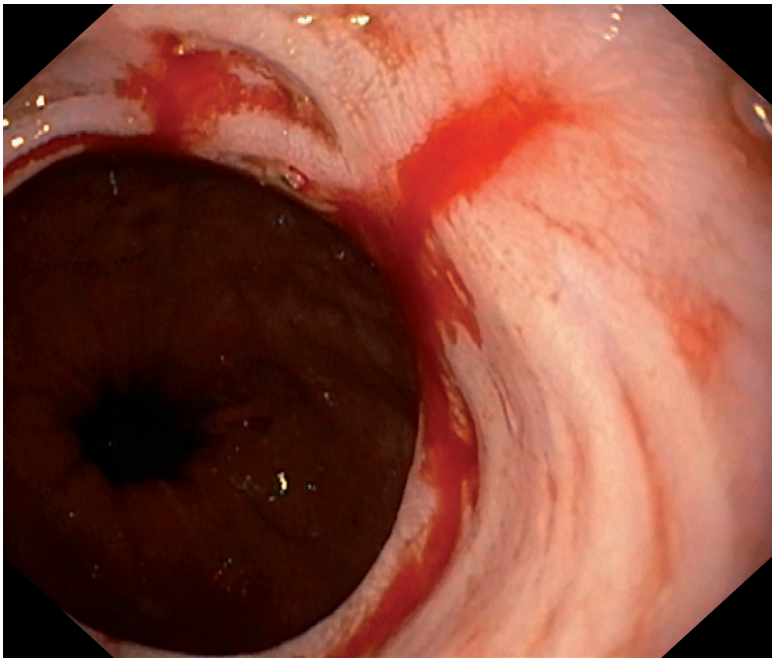


Fig. 4. Mallory-Weiss tear.

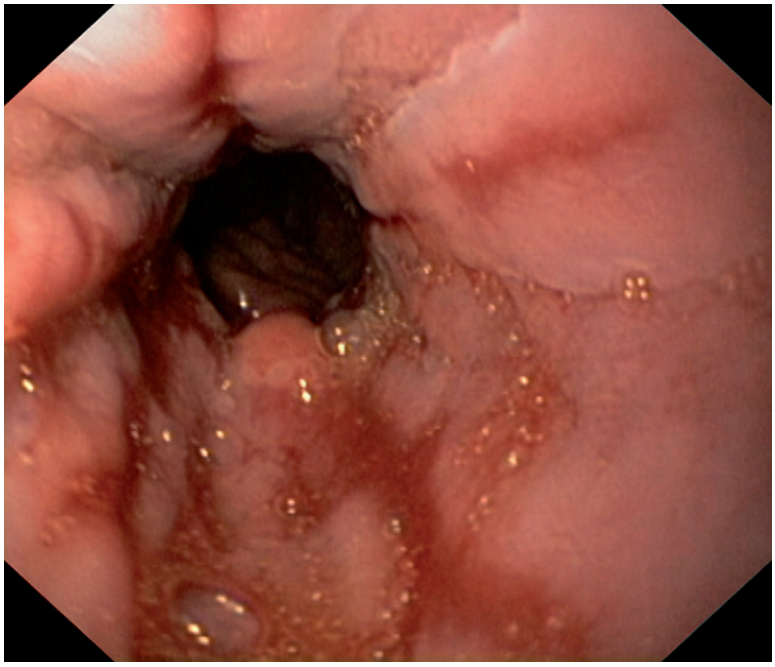


Fig. 5. Esophagitis.

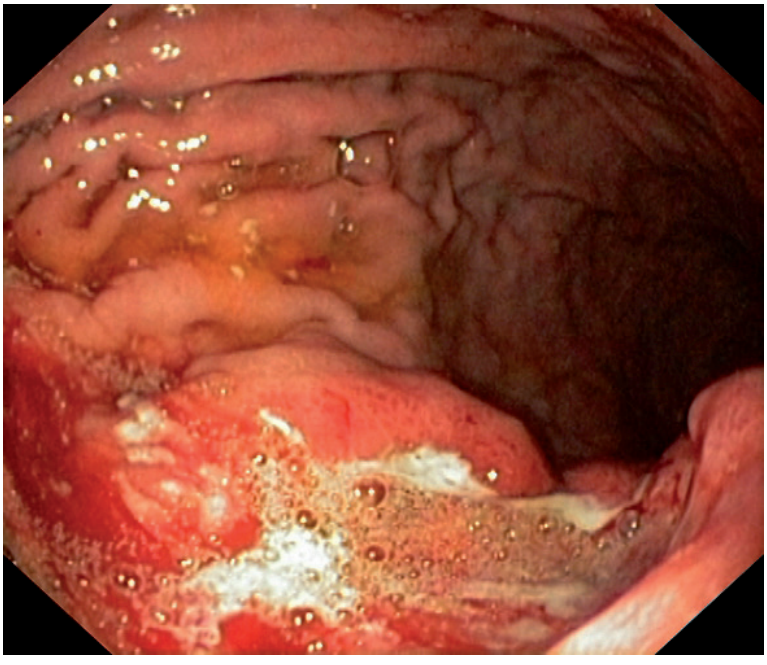


Fig. 6. Gastric cancer.

picture shows gastric wall tumor with foci of necrosis, partly covered with clots and fresh blood sipping underneath. The characteristic feature is the fragility of the mucosa and bleeding on contact with an endoscope.

Vascular malformations are disorders of vascular architecture, consisting of capillaries or small venous vessels. Arterial blood flowing through capillaries under high pressure may lead to their dilation and formation of haemangiomas, which may rupture. They are identified during routine endoscopy as a vivid red, flat or slightly elevated spots with irregular margins. If they bleed, a clot is formed on their surface, similar to the one observed in bleeding ulcer.

Another type of vascular malformation is superficially running submucosal artery, which under the influence of intense wall contractions can lacerate, leading to abundant hemorrhage. On endoscopy, a pulsating arterial blood flow can be seen, without any accompanying alterations on mucosal surface. This pathology referred to as Dieulafoy's lesion can occur in all parts of the GI tract, usually in the upper part of the stomach and in males.

DIAGNOSIS

Immediate initial clinical assessment of the patient's general condition and the possible resuscitation in hemodynamically unstable patients should be preceded by diagnostic proceedings [1, 8, 10, 11]. In case of massive vomiting patients may require endotracheal intubation in order to protect them against aspiration of bloody content. These patients require treatment in intensive care units, and further diagnostic tests to help determine the source of bleeding.

Relevant information is obtained from the patient such as upper abdominal pain, bloody or coffee ground vomiting, black tarry stools, rectal bleeding and pain in the chest. One must pay attention to medications taken by patients prior to admission, especially anticoagulants such as warfarin and clopidogrel, NSAIDs, aspirin and steroids, as they can adversely affect hemostasis. Patient's history in terms of digestive ulcers, previous GI bleeding, alcohol abuse, drugs, diabetes, atherosclerosis, chronic renal failure, chronic liver disease or obstructive lung disease are also important. During GI bleeding hemodynamic parameters may be normal, however, if its intensity grows, tachycardia and decrease of systolic blood pressure ensue. Orthostatic syncope may occur. Physical examination may reveal scars after previous abdominal surgery, stimulation of intestinal peristalsis, abdominal tension and signs of liver cirrhosis. Digital rectal examination is helpful to confirm the presence of black tarry stools or blood.

Basic laboratory tests should include complete blood count, levels of potassium, urea and creatinine, coagulation parameters and liver function tests [2, 8, 12–14]. Reduced red blood count, lower hemoglobin and hematocrit levels indicate anemia, what indirectly suggests that onset of bleeding was at least

12 hours prior to admission, and that the dynamics of the bleeding is not severe and adaptive mechanisms of the body help to refill the vascular bed. In addition to basic life support, treatment begins with crystalloids infusion, proton pump inhibitors given intravenously and blood transfusion if a decrease in hemoglobin concentration is less than 7 g/dL [2, 14, 15]. Blood transfusion is an unfavorable prognostic factor. The need for blood transfusion in the first 12 hours after admission to hospital doubles the risk of rebleeding and increases mortality up to 28% in patients bleeding from a peptic ulcer niche [2, 15].

NASOGASTRIC TUBE

A simple method to distinguish between bleeding from the upper and lower GI tract is still the insertion of the nasogastric tube in order to aspirate the gastric content. The presence of bloody or coffee-ground content confirms the diagnosis. However, it should be remembered that the lack of such a content in the tube does not exclude bleeding from the upper GI tract [2, 16].

ENDOSCOPY

Endoscopic examination of the esophagus, stomach and duodenum must be performed within 24 hours in hemodynamically stable patients, while in unstable patients as soon as possible [2, 7, 8, 10, 15–17].

In more than 90% of the cases endoscopy allows to locate the source of bleeding, assess its intensity, and stop it. To assess the intensity of bleeding a modified classification of Forrest is used for almost 40 years and differentiates active hemorrhage images from signs of recent hemorrhage. According to this classification stage Ia — active arterial bleeding, Ib — venous bleeding, IIa — visible vessel without active bleeding, IIb — adherent clot, IIc — flat pigmented hematin on ulcer base, III — lesions without signs of recent hemorrhage or fibrin-covered clean ulcer base. The intensity of bleeding classified as Ia, Ib and IIa requires prompt endoscopic treatment. The risk of rebleeding without the use of endoscopic hemostasis is almost 100% in Ia, 80% in Ib, 20–40% in II, and 3% in III [2, 7, 12, 14, 19–21] (Table 2). The introduction of endoscopic hemostasis techniques has led to reduction in mortality, rebleeding rates, hospital stay duration and number of emergency operations.

Administration of prokinetic drugs (erythromycin, metoclopramide) before endoscopic procedure accelerates gastric emptying making endoscopy easier to handle and thus reduces the need to perform repeated endoscopies in cases of diagnostic difficulties. The use of these drugs does not influence treatment outcomes [14, 22].

Table 2

Risk of peptic ulcer rebleeding with reference to endoscopic picture.

Endoscopic picture	Incidence	Rebleeding without endoscopic Therapy	Rebleeding with efficacious endoscopic therapy
Spurting (Forrest Ia)	12%	100%	15–30%
Oozing (Forrest Ib)	14%	80%	15–30%
Visible vessel (Forrest IIa)	22%	50%	5%
Adherent clot (Forrest IIb)	10%	33%	
Haematin covered flat spot (Forrest IIc)	10%	7%	
Clean bed of ulcer (Forrest III)	32%	3%	

It is possible to stop bleeding with a variety of endoscopic techniques that are further divided into thermal, chemical and mechanical modalities. Among thermal methods we can distinguish thermal laser photocoagulation and argon plasma coagulation as well as mono-, bi- and multipolar electrocoagulation, heat probe and microwave probe. Chemical methods include injection of vasoconstrictive agents, mainly epinephrine, sclerosants, thrombin and tissue adhesives. Mechanical methods include hemoclips, elastic endoscopic ligatures and loops. Presentation of all available techniques exceeds the size of this survey, however several of them will be discussed [2, 7, 14, 17, 19, 20–24].

Argon plasma coagulation (APC) is a contactless equivalent to monopolar coagulation, wherein argon gas is a carrier of electrical charge between the passive electrode attached to the patient externally and the active one supplying electric charge.

The transformation of electrical energy into heat occurs in place where the active electrode contacts with tissue, leading to localized coagulation. The argon, passed down an electrode catheter and energized with an intelligent-circuitry electrosurgical unit and patient plate, ionizes to produce a local plasma arc. The heating effect is inherently superficial (2–3 mm at most, unless current is applied in the same place for many seconds) because tissue coagulation increases resistance and causes the plasma arc to jump elsewhere. The distance from the end of the probe to the tissue is 2–10 mm. The dispersion of the electric charge occurs on a broad area like a spray. The depth of coagulation depends on the parameters of the generator (standard 60–90 W) and does not exceed 2–3 mm. This is particularly important in bleeding from the esophagus and duodenum, where the thin wall of the organ may perforate. The stream of electric charge finds the shortest way giving the possibility for tissue coagulation of places located laterally from the axis of the probe.

Inserting a probe ended with a steel needle through the channel of the endoscope permits submucosal injection of a sclerosant in bleeding site and surrounding area to obtain hemostasis. Epinephrine is most commonly used and applied in saline solution (ratio of 1 : 10,000). It evokes vasoconstrictive effect at the injection site, which is maintained for approximately 2 hours. Moreover, epinephrine increases platelet aggregation and locally compresses larger vessels via mechanical action. Epinephrine is applied in 1 mL aliquots around the base of the bleeding site, up to a total of 10–20 mL.

Hemostatic clips are also inserted through the channel of the endoscope. After identifying the bleeding vessel, a clip is opened, pressed into the vessel, and then clamped and disengaged from the probe. Clips are available in different clip arm lengths (6–10 mm). Metal clips cause mechanical pressure on the tissue and the vessel.

According to the latest available data, various endoscopic techniques must be associated with each other. Cochrane review of publications including 18 randomized trials on patients with bleeding peptic ulcers shows that an additional method of hemostasis combined with epinephrine solution injection reduces the incidence of rebleeding from 18.5 to 10 per cent and mortality from 4.7 to 2.5% [23–25].

The aforementioned methods are mainly used for endoscopic bleeding control regarding ulcerations. Most bleedings from mucosal tears in Mallory-Weiss cease spontaneously, but in the case of active bleeding, one of the endoscopic methods should be applied. In the event of bleeding from vascular malformations argon plasma coagulation and clips are applied most commonly. Dieulafoy's lesion with active hemorrhage can be controlled with clips or ligation. Neoplastic infiltration with bleeding, due to its size, is most often subjected to argon ablation. Because of its tissue friability other methods are usually ineffective [2, 7, 25].

The effectiveness of various endoscopic procedures is comparable and depends on the cause of bleeding, its intensity, general health status of the patient, and the experience of the physician. Initial hemostasis is achieved in 88–95% of patients. Approximately 10–20% of patients have early recurrence of bleeding requiring intervention, usually endoscopic. Endoscopic methods lead finally to bleeding cessation in 80–90% of patients with active hemorrhage [2, 7, 14, 19–25]. The primary disadvantage of endoscopic procedure is the possibility of tracheobronchial aspiration and perforation of the GI tract.

RADIOLOGY

Radiological diagnosis and treatment is used in cases where the endoscopic management alone is not effective. Angiography of the celiac trunk is essential and allows locating the site of bleeding and closing it. Vasopressin (0.2–0.4 U/30 minutes) is injected via angiographic catheter that is positioned in close proximity to the

bleeding site and then angiographic control is performed. If a bleeding advances, vasopressin infusion must be applied for 6–12 hours. In order to counteract the side effects of systemic vasopressin action, a simultaneous nitroglycerin infusion is recommended. Vasopressin infusion is effective in 50–80% of patients; however, nearly 50% have early recurrence of bleeding. Another method is highly selective embolization of the bleeding vessel with embolic agent. Embolic agents include temporary materials such as gelatin sponge (e.g., Gelfoam), oxidized cellulose or polyvinyl alcohol. Complications such as mesenteric artery thrombosis and intestinal wall ischemia occur in about 3% of patients. The effectiveness of radiological methods for bleeding control is technically 92–100%, but only 50–94% clinically. Rebleeding occurs in 9–47% of patients. The drawbacks of arteriography are the possibility of kidney damage by the administered contrast agent, allergic reaction to injected agent, exposure to radiation, and bleeding from the site of vascular access [2, 14, 25].

SURGERY

The ineffectiveness of the above mentioned methods, symptoms of bleeding, and situations where the patient requires more than 6 units of blood to be transfused are indication for prompt surgical treatment. The emergency surgery rate for peptic ulcer bleeding control has decreased in recent years, and according to the British data has fallen from 8% in 1993 to 2% in 2006, and according to the US data from 21% to 9%, respectively [4, 5].

The course of operation depends on the cause of bleeding, patient's general condition, the mode of performed surgery and the surgeon's experience. Surgery may be impeded by excessive air filling of the intestines and the possibility of iatrogenic damage to the mucosa after endoscopic procedure and coagulopathy after numerous blood transfusions.

The bleeding peptic ulcer in the stomach is usually treated surgically by partial gastric resection with the ulcer niche. Restoration of gastrointestinal continuity depends on the anatomical conditions and aims to restore them as closely as possible. Surgical control of bleeding from duodenal ulcer niche involves duodenotomy and undersewing of a bleeding vessel, followed by pyloroplasty. Sometimes, in case of extensive ulcerations involving pylorus, antrectomy is performed. In the era of proton pump inhibitors transection of vagal trunks under the diaphragm is not a recommended procedure, whereas in the post-operative management it is advisable to block the gastric secretion.

Bleeding from the neoplastic infiltration may require surgical treatment. It involves the removal of all or part of the stomach and a cancerous tumor with disease-free margins and subsequent restoration of the continuity of the gastrointestinal tract. Efficacy of bleeding cessation after surgery is 70–100%. Recurrence

of bleeding occurs in less than 5% of patients treated surgically, but the perioperative mortality rate is between 10 and 50% [2, 14, 25].

PHARMACOTHERAPY

Targeted pharmacotherapy is essential in the treatment of bleeding from upper GI tract. Patients with symptoms of bleeding should be given 80 mg pantoprazole, omeprazole or esomeprazole intravenously (bolus), followed by continued infusion at a dose of 8 mg/hour until the endoscopic diagnosis of the bleeding source is made and in case of its confirmation continued until 72 hours after the endoscopic bleeding cessation [2, 14, 25, 26]. Cochrane systematic review of literature on the basis of 6 randomized clinical trials showed no statistical difference in the mortality rate, recurrent bleedings and frequency of emergency surgical procedures after initial use of PPI relative to the control group (placebo or histamine receptor blockers). However, patients receiving PPI rarely had a history of endoscopic signs of bleeding (37.2% vs. 46.5%) (OR = 0.67) and required fewer endoscopic controls (8.6% vs 11.7%) (OR = 0.68) [27].

RECURRENT BLEEDING

Symptoms of bleeding after endoscopic examination or treatment and hemodynamic disturbances suggest recurrence of bleeding. Rebleeding is more frequent in patients in whom active bleeding was observed on endoscopy, in patients aged over 60 years, manifesting symptoms of shock on admission, with a baseline hemoglobin level below 10 g/dL. Diameter 20 mm and above of the ulcer niche or multiple ulcers are factors that predispose to rebleeding [2, 13, 14, 25]. Large ulcers on the posterior wall of the duodenal bulb and the lesser curvature of the stomach can penetrate down to gastroduodenal or left gastric artery, making endoscopic treatment ineffective.

Rockall risk scoring system attempts to identify patients at risk of adverse outcome following acute upper gastrointestinal bleeding (Table 3). Rockall risk scoring system has been widely used mainly in the US for nearly 20 years [12]. This scoring system uses clinical criteria (increasing age, co-morbidity, shock) as well as endoscopic finding (diagnosis, stigmata of acute bleeding). According to this score, patients with no evidence of active bleeding with a low risk of recurrent bleeding may leave the hospital on the day of diagnosis, and patients with a high risk of recurrence of bleeding should undergo endoscopy inspection next day.

In Europe, the more popular Glasgow-Blatchford bleeding score (GBS) takes into account the level of urea in the blood, hemoglobin, systolic blood pressure, heart rate, presence of tarry stools and syncope, positive medical history of liver

Table 3

Rockall risk scoring system for assessment after an episode of acute upper GI bleeding [12].

Age	Younger than 60 years	60 to 79 years	80 years or older	—
Shock symptoms, systolic Blood pressure, heart rate	Shock absent, blood pressure 100 mm Hg or greater, heart rate 100 bpm or greater	Tachycardia, blood pressure 100 mm Hg or greater, heart rate 100 bpm or greater	Hypotension, blood pressure less than 100 mm Hg	—
Comorbidities	No major comorbidity	—	Heart failure, coronary artery disease, any major comorbidity	Renal failure, liver failure, disseminated malignancy
Endoscopic diagnosis	Mallory-Weiss tear or no lesion identified, and no stigmata of recent hemorrhage	All other diagnoses	Malignancy of upper GI tact	—
Stigmata of recent hemorrhage	None or dark spot only	—	Blood in upper GI tract, adherent clot, visible or spurting vessel	—

Risk of rebleeding and mortality based on Rockall Risk Score:

	Score								
Risk	0	1	2	3	4	5	6	7	>8
Rebleeding (%)	4.9	3.4	5.3	11.2	14.1	24.1	32.9	43.8	41.8
Mortality (%)	0	0	0.2	2.9	5.3	10.8	17.3	27.0	41.1

bpm = beats per minute

failure and coronary heart disease [13] (Table 4). GBS is able to determine more precisely the patients who may require re-intervention, particularly those in which the dynamics of bleeding was not high. Based on this scale 16–25% of patients with symptoms of bleeding can be early discharged from hospital. Recurrence of bleeding is an indication to re-take endoscopic treatment. In case of endoscopic treatment failure it is recommended to perform angiography combined with embolization. Surgical treatment for bleeding peptic ulcer niche is used as a last resort [2, 14, 20, 25].

Patients who manifest signs of bleeding and in case of failure to identify the source of bleeding, the endocapsule or enteroscopy should be used for small bowel

Table 4

Blatchford scoring: admission risk markers and associated score component values [13].

Admission risk marker	Score component value
Blood urea, mmol/L	
6.5–≤8	2
8.0–<10.0	3
10.0–<25	4
≥25	6
Hemoglobin for men, g/dl	
12.0–13.0	1
10.0–<12.0	3
<10.0	6
Hemoglobin for women, g/dl	
10.0–<12.0	1
10.0	6
Systolic blood pressure, mm Hg	
100–109	1
90–99	2
<90	3
Other markers	
Pulse ≥100/min	1
Presentation with melena	1
Presentation with syncope	2
Hepatic disease	2
Cardiac failure	2

examination [2, 14]. Examination with endocapsule is non-invasive and it does not require sedation and in most cases permits to evaluate the whole intestine. The main drawback of this method is the inability to stop bleeding.

Enteroscopy allows the exact location of a bleeding source and using one of the endoscopic methods to stop it, and in case of failure tattoo marking place before surgery. The effectiveness of this treatment depends mainly on the physician's experience in endoscopic procedures. It is often hard to visualize the entire small intestine, the treatment requires a special, rarely available instrumentation, perforation of the GI tract or damage to the mesentery of the small intestine may occur during the examination.

A proper selection of patients who can be safely discharged from the hospital is a major problem. These are patients below 60 years of age, hemodynamically stable,

without comorbidities, without coagulation disorders, with a hemoglobin level of greater than 8 g/dL, in whom bleeding occurred outside the hospital, and there was no active bleeding on endoscopy. They must be patients whose consciousness and material status can afford home treatment [2, 20, 25] (Table 5).

Table 5

Selection criteria for early hospital discharge (following early endoscopy) [1, 20, 21].

Age <60 years
No hemodynamic instability
No severe coexisting illness
A hemoglobin level over 8.0 g/dl after adequate vascular volume expansion
Normal coagulation studies
Onset of bleeding outside the hospital
Presence of a clean-based bleeding lesion
Adequate social support at home with an ability to return promptly to healthcare facility

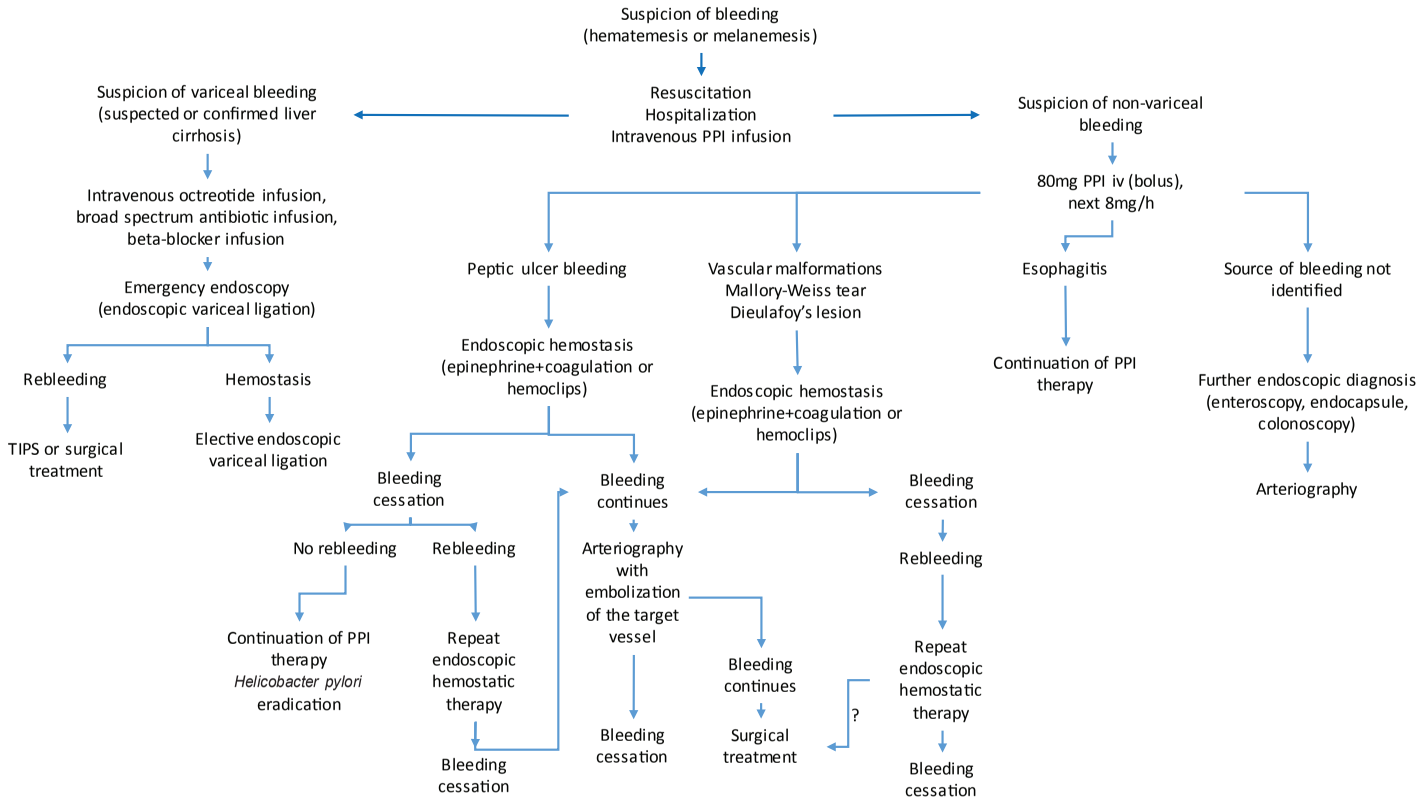
Table 6

Endoscopic principles of management in nonvariceal upper GI bleeding [1, 20, 21].

Endoscopy should be performed within 24 hours that follow presentation when possible.
All patients exhibiting high-risk endoscopic stigmata should receive endoscopic hemostasis.
Highly selected patients at very low risk for rebleeding may be discharged home immediately after an early endoscopy.
Patients exhibiting a pigmented dot or clean base ulcer do not require endoscopic hemostasis.
Although better than doing nothing, epinephrine injection alone provides suboptimal efficacy and should be used in combination with another modality such as clips, thermal or sclerosant injection that are also efficacious alone.
Endoscopists should opt for an efficacious hemostatic method, with which they are more comfortable.
Finding a clot in an ulcer bed requires an attempt at dislodgement in order to treat the underlying lesion.
Optimal treatment of adherent clots remains controversial; endoscopic therapy should be considered but intensive PPI therapy may be sufficient.
Second-look endoscopy should be considered on a case-by-case basis if the effectiveness of endoscopic hemostasis is in question or for patients at particularly high risk of rebleeding.

BLEEDING PROPHYLAXIS

Helicobacter pylori infection and NSAIDs are the factors contributing to the occurrence of bleeding peptic ulcerations. Eradication of bacteria, avoiding NSAIDs, aspirin and clopidogrel reduces the possibility of bleeding. Eradication of *Helicobacter pylori* reduces the risk of distant recurrence of bleeding from 20% to 2.9%.



Algorithm for the management of acute upper gastrointestinal bleeding.
TIPS — transjugular intrahepatic portosystemic shunt, PPI — proton pump inhibitor

Effectiveness of eradication should be confirmed after treatment. Patients requiring anticoagulant medications for cardiac and vascular reasons should take them under cover of PPI [2, 4, 9, 25].

BLEEDING RELATED TO PORTAL HYPERTENSION

The risk of bleeding increases when the portal-hepatic venous pressure gradient exceeds 12 mm Hg. Bleeding affects about 40–80% patients with liver cirrhosis and portal hypertension and the 6-week mortality rate after the first hemorrhage is about 50% [10, 14].

90% of patients with liver cirrhosis lasting 10 years or more bleed from esophageal varices. Esophageal variceal hemorrhage occurs most commonly, however in more than 10% of patients varices are located in the gastric fundus. Patients with large varices, covered with numerous red spots on the mucosa and decompensated cirrhosis (Child-Pugh class C) may bleed into the lumen of GI tract in more than 70% of cases. It was observed that in nearly 25% of patients with cirrhosis and esophageal varices bleeding is caused by other pathology (usually peptic ulcer disease), and further 25% of patients present other lesions on endoscopy that may be the bleeding source [2].

Initial management consists of fluid resuscitation and evaluating the possibility of liver failure. It is proposed to start the treatment from administration of synthetic somatostatin longer-acting analogue octreotide, causing selective visceral vasoconstriction. Octreotide is administered by intravenous infusion at a dose of 1–2 μg/kg/hour and applied for 3–5 days. Reducing the visceral flow via administration of beta-blockers in case of active bleeding as an isolated procedure is not effective, but their use in patients after elective repeated endoscopic procedures reduces the rate of rebleeding. It is also desirable to provide vitamin K and fresh frozen plasma in order to improve blood coagulation. Endoscopy is performed immediately, preferably up to 12 hours from the onset of hemorrhage. The procedure is significantly hindered by massive blood pouring out of one or more varices and shutting out the view of the endoscope. Endoscopic variceal ligation has been shown to be more efficacious than sclerotherapy. Rubber band ligation is performed on several levels, as well as injection of a vasoconstrictive agent. The efficacy of endoscopic cessation of bleeding is assessed at 75–95%, but more than 30% of patients suffer from recurrent bleeding. Because infections are common in patients with bleeding varices, prophylactic antibiotics are initiated. They have been shown to decrease the infection rate by more than 50%, decrease rebleeding, and improve survival [14, 28].

If a temporary bleeding cessation is achieved, endoscopic procedures are repeated several times in a planned manner at 2 week intervals. Reduction of rebleeding rate develops after 3–4 sessions.

If endoscopic methods are not effective, it is necessary to perform mechanical tamponade. Tamponade is made using special probes with two filled balloons (gastric and esophageal) and the suction channel for gastric contents (Sengstaken-Blakemore probe). The effectiveness of this method in immediate cessation of bleeding is 94% but recurrent hemorrhage occurs in 60% of patients after balloon deflation. The probe can be kept inflated in the esophagus up to 24 hours.

SUMMARY

In the last few years treatment tactic of upper GI bleeding changed radically. The combination of endoscopy and pharmacotherapy reduced mortality in patients suffering from acute GI hemorrhage. Endoscopy performed early, up to 24 hours permits to recognize the source of bleeding, stop it and identify the patients more vulnerable to the risk of recurrent bleeding. The use of multiple endoscopic methods for bleeding control together with eradication of *Helicobacter pylori* and high doses of PPI decreased the recurrence rate of bleeding along with the amount of emergency surgical procedures. Strict indications for blood transfusion had a positive impact on the outcomes of treatment. Future research will focus mainly on bleeding prevention and on the treatment of elderly patients, receiving anticoagulants and anti–inflammatory drugs.

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